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Twenty-third Annual Meeting, held at St. John's Parish Hall Washington, D.C., May 4th, 5th, and 6th, 1897.

The President, Dr. M. A. Starr, in the chair.

Dr. John Jenks Thomas, of Boston, presented a paper, entitled:

TWO CASES OF ACUTE ASCENDING PARALYSIS, WITH AUTOPSIES.

The first case was that of a woman, thirty-six years of age, who entered the Boston City Hospital, August 29th, 1896. She noticed a weakness of the legs, beginning suddenly on Aug. 5th, which became a complete paralysis the next day. The legs felt numb and dead, and were somewhat painful to pressure. There was retention of urine. On examination on entrance into the hospital the legs were paralyzed, except that the toes and feet could be moved slightly; the patellar and plantar reflexes were absent, and sensation was not affected. The arms were also paretic. There was no tenderness in the upper extremities. The internal organs were normal. The area of splenic dulness was not increased. Later there was bronchial breathing in the left back, and considerable dyspnea. The urine showed a trace of albumin and was neutral in reaction. The specific gravity was 1,018. A few questionable granular casts and a little blood were found in the sediment. The paralysis of the extremities increased, and there were incontinence of urine and dyspnea. The patient died on August 24th. The temperature varied from 100.5 deg. to 102.0 deg. F. at first; later from 98.4 deg. to 100.0 deg.

The autopsy showed a bronchopneumonia of the left lung; chronic diffuse nephritis; arteriosclerosis of the aorta; myoma of the uterus, and an acute anterior poliomyelitis.

The cultures from the heart, liver and spleen were sterile. Those from the kidney gave colon bacillus and a liquefying organism, and those from the spinal cord also showed liquefying organisms (putrefactive organisms).The microscopic examination of the spinal cord showed, first, acute inflammatory exudation in the anterior horns of the gray matter, with parenchymatous degeneration of the nerve cells and processes; secondly, infiltration of the perivascular lymph spaces and dilatation of the vessels of the anterior horns; thirdly, moderate infiltration about the vessels of the posterior horns and of the white matter of the cord; fourthly, slight parenchymatous degeneration of the nerve fibres of the white matter of the cord; fifthly, slight degeneration of the posterior nerve roots and marked degeneration of the anterior nerve roots; sixthly, parenchymatous degeneration and perivascular infiltration of the peripheral nerves; seventhly, absence of micro-organisms in the sections. The brain could not be obtained for examination.

The second case was of a man, aged thirty-five, who entered the Boston City Hospital on Oct. 5th, 1896. He had been well up to twelve days before entrance, at which time he began to complain of numbness and weakness in his legs. He had had very little pain, but was said to have had one attack of dyspnea before entrance. He ascribed his illness to exposure to cold and rain. On entrance there was marked paralysis of both legs, both arms and of the muscles of the neck. The tendon reflexes were lost. The plantar reflexes were present, but diminished. The cremasteric and abdominal reflexes were normal. Sensation was normal, and there was no tenderness. Urine was acid and without albumin and sugar; the sp. gr. was 1,025. The patient had an attack of dyspnea on August 7th, in which he died. The temperature was normal.

The autopsy showed bronchopneumonia, and hemorrhage of the lungs, enlargement of the spleen, congestion of the kidneys, congestion of the brain and cord, parenchymatous degeneration of the peripheral nerves and of the motor cells of the spinal cord.

Cultures from the heart, liver, spleen, kidneys and meninges of brain and spinal cord were sterile. Four cultures were taken from the brain, three from the meninges of the brain, and one from the spinal cord. Three of these (two from the brain and one from the meninges) showed no growth. Dilution and replants from the others showed nothing but saprophytes (a spore-forming bacillus), a large coccus, and a large diplococcus.

The microscopical examination showed: first, parenchymatous degeneration of the peripheral nerves, varying in extent, and present to a greater or less degree in all the nerves examined; secondly, degenerative changes in all the large ganglion cells of the anterior horns of the cord, with destruction and fragmentation of the protoplasmic granules and loss of the nuclei of the cells; thirdly, the nerve cells of other portions of the gray matter of the cord, oblongata, brain and spinal ganglia unchanged; fourthly, no change of the white matter of the cord; fifthly, absence of micro-organisms in the tissues.

The author concludes that acute ascending paralysis is a clinical collection of symptoms arising from an affection of the primary motor neuron, probably toxic in nature (but not due to a distinct micro-organism), which varies in character in different cases, consisting in the milder forms of a degeneration of the motor cells and processes with disappearance of the chromophilic granules of the protoplasm and loss of the nucleus and nucleolus; and in the severer forms amounting to an acute inflammatory process of the anterior horns of the gray matter with infiltration of the perivascular lymph spaces and degeneration and destruction of the motor cells and processes.